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PHYSIOLOGICAL REACTIONS OF THE HUMAN ORGANISM TO TRANSVERSE ACCELERATIONS AND MEANS OF RAISING THE RESISTANCE TO SUCH FORCES

by A. S. Barer, et al.

Paper presented at the XV International Astronautical Congress, Warsaw, September 7-12, 1964

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PHYSIOLOGICAL REACTIONS OF THE HUMAN ORGANISM TO TRANSVERSE ACCELERATIONS AND MEANS OF RAISING THE RESISTANCE TO SUCH FORCES

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Translation of "Fiziologicheskiye reaktsii organizma cheloveka na poperechno napravlennyye uskoreniya i nekotoryye puti povysheniya ustoychivosti k etim vozdeystviyam"

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PHYSIOLOGICAL REACTIONS OF THE HUMAN ORGANISM TO TRANSVERSE ACCELERATIONS AND MEANS OF RAISING THE RESISTANCE TO SUCH FORCES

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Any flight aboard a spacecraft is associated with the action of various types of acceleration on the astronaut. Accelerations of long durations arise during the ascent and penetration of the craft into the dense layers of the atmosphere and also during the maneuvering of the craft in the course of flight.

To date, after the first successful orbital flights, considerable data have, of course, accumulated concerning the effect of acceleration on the organism of man and animals (L. Buhrlen, 1937, H. G. Armstrong and I. W. Heim, 1938, O. Gauer and S. Ruff, 1939, E. R. Ballinger, 1952, A. R. Kotovskaya, and others). However, a whole series of problems remains relatively unstudied. Thus, there are only isolated reports on the study of the endurance limits (N. C. Clarke, 1959, A. S. Barer, 1962), and on the increase of the resistance of the human organism to this type of force. Clarke notes that the optimum tilt angle of the back of the pilot's chair is 25°, i.e., 65° to the acceleration vector. The author plotted a graph of man's resistance to this force. The maximum acceleration studied in this work was 12 G for a duration of 5 sec. It is obvious that such a low time endurance restricted any further investigations. In Clarke's subsequent works, and also in the works of other investigators (F. W. Zechman, 1959, A. Slonim, 1962, G. C. Mueller, 1961, I. F. Watson, and N. Cherniack, 1962, E. F. Lindberg and others, 1962) recommendations were given for an angle of 12°, i.e., 78° between the acceleration vector and the longitudinal axis of the human body.

Our work (1961-1962) was carried out in three stages. The first stage was devoted to the study of human endurance limits to prolonged accelerations acting in the back-chest direction at 65° angle to the longitudinal body axis (A. S. Barer et al., 1962-1963). The second stage consisted of a study of various means of raising man's resistance to transverse accelerations. The results of these experiments determined the third stage of the investigations, in which the resistance to accelerations with respect to time was examined under optimum conditions.

The work was conducted with a large-radius centrifuge. During the experiments, recordings were made of the EKG, arterial pressure, volume indices of external respiration, EMG, EEG, acuity and field of vision; also the working capacity was investigated. In some of the experiments, X-rays of the chest organs were taken and a gas analysis of the exhaled air was performed.

The ECG was recorded continuously in three Neb leads during the action of acceleration and during various periods of the aftereffect. The arterial pressure was measured in the brachial artery by Korotkov's method. The tones and pressure in the cuff were recorded by means of remote transmission. The indices of external respiration were recorded with a pneumotachograph. An EMG recording was made in unipolar leads from intercostal muscles, the sternocleidomastoid muscle, the externus obliquus abdominis muscle, and the quadriceps femoris muscle. The EEG was recorded in four unipolar leads (frontal, temporal, parietal, and occipital from the right hemisphere) and in one bipolar temporal-occipital lead from the left hemisphere with an EG-138 electroencephalograph with an EA-101 frequency analyzer and integrator. The vision field was studied by means of a horizontal electrical perimeter, and the visual acuity with the aid of Landoldt rings (using P. M. Suvorov's method). The working capacity was studied with a special "test-pilot" instrument. The X-raying was carried out in the form of lateral left pictures, before, during, and after the action during the phase of deep inhalation (up to 12 G). In experiments where the endurance limits were studied, the centrifuge was stopped upon command by the subject. During the experiments, the subjects were under close medical supervision.

In all, about 500 experiments involving participation of 70 subjects were conducted. The established endurance limits are shown in Figure 1. The maximum acceleration during the first stage of the experiments was 16 G. The discontinuation of further experiments was caused by the low resistance of man to these values of acceleration. Thus, for example, at 14-16 G, unconsciousness occurred in a number of cases after only 5-30 sec. It was found that the chief limiting factors were impairments in the circulatory and respiratory systems. Furthermore, impairments in blood circulation were primarily associated with the action of inertial forces whose direction coincided with the longitudinal body axis.

Thus, in acceleration at an angle of 65° to the longitudinal body axis, the component along the pelvis-head axis was 42°. Knowing that man's endurance to acceleration is directed along this axis and using theoretical arguments only, we can readily understand that only if this aspect of the problem is examined (which is possible only with considerable qualifications), the endurance limit beyond which visual and brain disturbances are certain to take place is in the range of 14-16 G, when the component along this axis reaches 6-7 G. As is known, this phenomenon is primarily due to hemodynamic shifts which cause an impairment of the blood circulation in major organs, particularly in the brain. In addition, it should be assumed that substantial difficulties in the system of external respiration (due to the effect of inertial forces) cause additional difficulties in the oxygen supply.

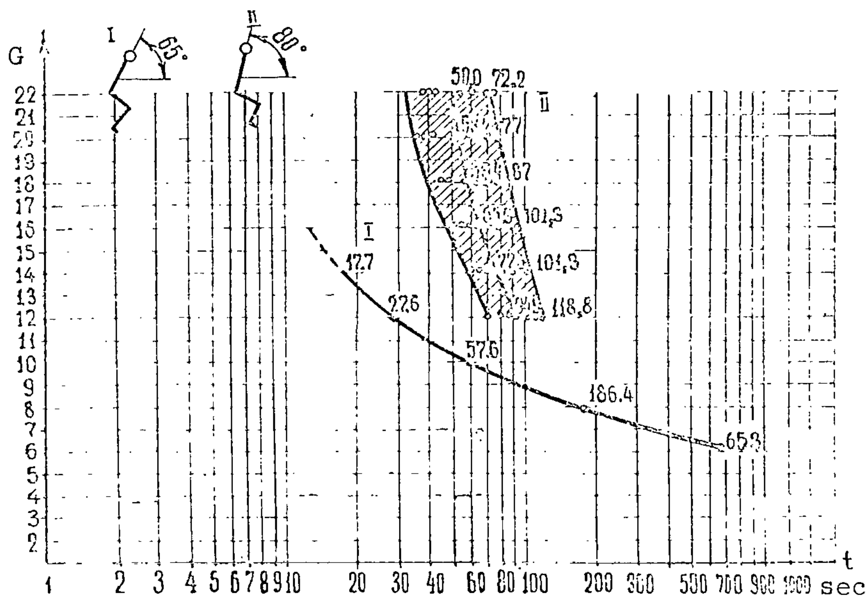

Figure 1. Graph of endurance to accelerations acting in the back-chest direction

Calculation shows that a further increase (from 65^o up) in the angle between the direction of acceleration and the longitudinal body axis is accompanied by a substantial decrease in the component along the pelvis-head axis and a relatively slight increase of the component along the back-chest axis, which is undoubtedly more advantageous from the physiological standpoint. Thus, when the acceleration acts at an angle close to 80^o,[1] the component along the pelvis-head axis decreases more than twofold as compared to its value during the action of acceleration at a 65^o angle, while the component along the back-chest axis increases by only 7 percent. This was one of the major reasons for the continuation of experiments with this direction of acceleration.

In the second series of experiments, an attempt was made to evaluate the various means of raising human resistance to acceleration directed

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[1]An exact observation of the angle between the acceleration vector and the longitudinal axis of the human body is sometimes difficult because of individual anatomical differences.

along the back-chest axis. The investigation included: various tilt angles of the human body with respect to the acceleration vector, breathing with oxygen at atmospheric pressure and with oxygen and air at excess pressure, and the use of tiltable chairs. It was established that the optimum conditions are created when the angle between the total acceleration vector and the longitudinal body axis approaches 80°. The 90° angle was rejected because of the appearance, under these conditions, of sensations of acute pain in the afterbreast and the epigastrium, relative bradycardia and arrhythmia of the cardiac activity, and deep shifts in the external respiration indices arising as low as 8-12 G.

At times, a cessation of breathing was observed following the painful sensations. On the basis of direct measurements of the volume indices of external respiration carried out under these conditions and X-ray investigations, a sharp decrease in the depth of breathing during the flattening of the chest and a rise of the rear section of the diaphragm were noted (Figure 2). Thus, for example, the area of the pulmonary field at 12 G was 65 percent of the original value.

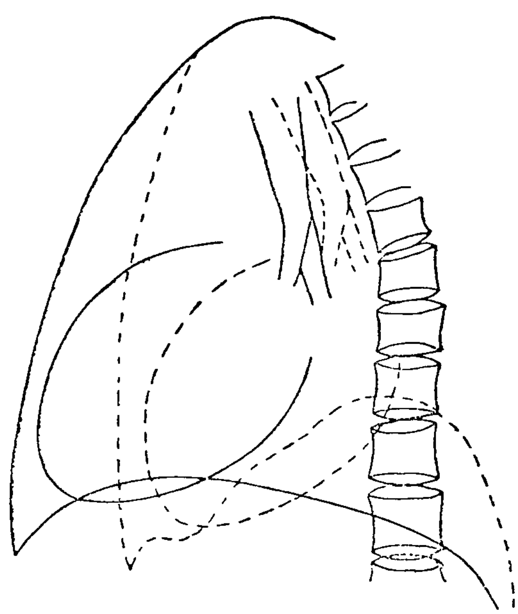

Figure 2. Diagram of X-ray changes in human chest during the action of transverse accelerations:
——— background
– – – – acceleration of 12 G, 80° angle

At an acceleration of 12 G directed at an angle of 80°, as compared to the acceleration of 65°, a 2-3-fold increase in the time endurance to this force was noted. Respiration with oxygen at atmospheric or slightly higher pressure (whose magnitude should be selected) considerably improved the morale of the subjects. The responses of the organism were more stable, and the time endurance grew by an additional factor of 1.5-2. It was noted in the same experiments that tilting of the chair also has a favorable effect on human resistance to acceleration. This resistance is expressed by a diminution in painful sensations and petechial subcutaneous blood effusions, and also by a facilitation (as evaluated subjectively) of the breathing movements.

Experimental conditions where the tilt angle of the back of the chair was 80°, where the subjects breathed with oxygen or air at atmospheric pressure, and where a tilting chair was used were adopted in the third series of experiments.

An inspection of the graph (Figure 1) makes it immediately apparent that the measures which were taken proved to be highly effective and made it possible to perform the experiments with an acceleration of 22 G at an average duration of 50 sec. Later, experiments were performed with an acceleration up to 26.5 G at a predetermined time of 8 sec.

According to subjective estimates, the chief limiting factor in these experiments was the sensation of hindered breathing and general fatigue. In contrast to the experiments involving an acceleration at a 65° angle, there was not a single case of unconsciousness at higher accelerations in a fairly large number of experiments. The subjects tested felt much better after 20 G than after acceleration of 14-16 G acting at an angle 65°.

It should be kept in mind, however, that if such a high acceleration acts long enough (a minute or more), then against the background of the apparent well-being in the course of the action itself (based on subjective and objective data), during the following aftereffect, symptoms may appear in certain cases which indicate the presence of extravasation in the pulmonary tissue. Such phenomena were also observed by American investigators (J. Watson and M. Cherniack, 1962). In our view, this fact should be of particular interest to specialists working on training systems. Of interest in this connection are experiments on animals (I. M. Kazem, B. M. Kogan, A. S. Barer, 1963) where it was shown that frequently repeated accelerations result in extensive morphological changes in many systems and organs, including pulmonary tissues. However, when the same magnitudes of acceleration were used, but with week-long intervals between the experiments, the morphological changes were reduced to a minimum.

Of considerable interest are the changes in the basic functional systems of the organs. The reaction of the cardiovascular system amounted mainly to the following. The acceleration always produced sinus tachycardia whose degree depended on the magnitude of the acceleration.

As a rule, the tachycardia appeared even before the start of the acceleration (emotional factor). As the rotation started, the frequency of the heartbeat gradually increased and reached a maximum for the given experiment at the end of the acceleration or at the start of the plateau.[1] In many experiments (under the influence of accelerations of 4-8 G at an angle of 65^{o}, and an acceleration of 12 G acting at an angle of 80^{o}), after the first 10-30 sec of the plateau, a certain decrease in the heartbeat was observed (by 10-20 heartbeats per minute). This lower level lasted until the end of this period. For a 10-12 G acceleration at an angle of 65^{o}, the heartbeat reached maximum values (170-185 beats per minute). A further increase in acceleration no longer caused an increase in the frequency of the heartbeat above the indicated values.

When the acceleration was acting at an angle of 80^{o} to the longitudinal body axis, the reaction of the cardiovascular system was less strained than in experiments where the acceleration was applied at 65^{o}, and this was expressed, in particular, by a lesser increase of the heartbeat frequency. In this case as well, no further increase in this value was observed after 12 G. The level of the heartbeat frequency during the period of the plateau usually remained fairly stable.

The functional state of the heart could be evaluated to some extent by means of the electrocardiographic indices. An increase in the heartbeat frequency was usually accompanied by a blending of the P and T waves, an increase in the systolic index, a decrease in the voltage of the waves of the complex QRS and T, and, at certain times, by a decrease of the S-T interval.

The P-Q interval was sometimes shortened to 0.1 sec. The QRST interval, i.e., the electric systole of the heart, changed as a function of the heartbeat frequency. However, as the heartbeat frequency increased, the shortening of this interval was relatively smaller than that of the R-R interval, and for this reason the systolic index (according to Fogel'son-Chernogorov) rose steadily and reached considerable values (70-80 percent for an initial value of 45-50 percent).

Major changes were noted in the voltage of the waves of the ventricular complex, particularly RT. As the acceleration increased, the voltage of the RT waves decreased steadily in all the leads, and most appreciably in the frontal lead. During the slowing down of the centrifuge and the following aftereffect, the voltage of these waves increased considerably, and the T-wave occasionally exceeded the initial level by a factor of 3-5 (Figure 3).

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[1]By plateau we refer to the period of time when the given acceleration is maintained without change.

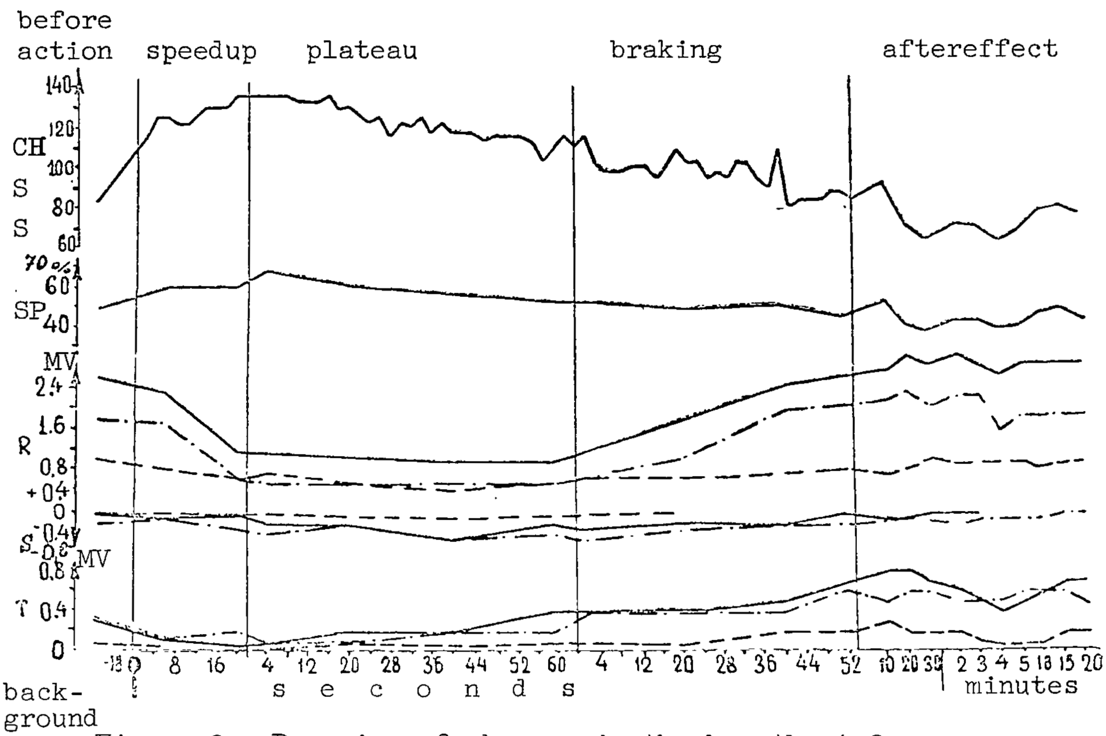

Figure 3. Dynamics of changes in the heartbeat frequency, systolic index, and voltage of RS and T waves. EKG for a 20 G acceleration at 80° angle

——————— voltage of waves in Neb's front lead

— — — — — in hind lead

—·—·—·—·— in lower lead

The lateral X-ray pictures showed a flattening of the anterior contour of the heart, a decrease in the density of the cardiac shadow, and a displacement of the heart in the dorsal direction. These changes progressed steadily with the increase in acceleration and were greatest in experiments with acceleration acting at a 90° angle. In addition, a displacement of the apex of the heart from the initial level was observed in a majority of cases. This phenomenon was most pronounced in experiments involving acceleration at angles of 65° and 80°, and was also observed, although to a lesser extent, when the inertial forces were acting in a strictly transverse direction.

The above-described displacements of the heart were positively reflected in the voltage of the ECG waves. However, the recorded changes of the voltage of the ECG waves cannot be explained by these displacements alone.

Changes in the bioelectrical activity of the myocardium during the influence of acceleration and in the course of the period of the aftereffect may be due to a whole set of factors: hypoxia, change in the tone of the autonomic nervous and pituitary-adrenal systems shifts in the ionic relationships, variation in the filling of the heart chambers with blood, etc.

Impairments in the rhythmicity of the heartbeat were observed fairly often in the form of extrasystoles of various origins and in phasic sinus arrhythmia. The extrasystoles were most often single and were encountered only during the influence of acceleration in the course of the plateau. Multiple ventricular extrasystoles of the bigeminal type were recorded in two cases at an acceleration of 8 G. At times, the appearance of extrasystoles was distinctly related to the breathing act. In certain cases, the extrasystoles appeared in response to some verbal order of the experimenter, which indicated the emotional cause of the phenomenon. After an acceleration of over 10 G, a respiratory phasic sinus arrhythmia would often appear.

The arterial pressure in the brachial artery during the action of accelerations at an angle of 65° changed in the following manner. Both the systolic and the diastolic pressure increased more appreciably; the greater was the magnitude of the acceleration. Already at 8 G, the systolic pressure reached 220, and the diastolic pressure, 170 mm Hg. The pulse pressure at 4 and 6 G increased, and at higher values of the acceleration decreased to the initial level. During the first minute after the centrifuge came to a halt, there was always observed an increase in the pulse pressure due to a rise in the systolic pressure. The degree of this increase was directly related to the magnitude and duration of the effect, and after experiments involving acceleration at a 65° angle, the pulse pressure was higher than after experiments in which the acceleration was applied at 80°, and could reach appreciable values (100-120 mm Hg). It is known that the rise in pulse pressure is an indirect indication of an increased stroke volume of the blood (Wiggers, 1957). In our review, the increased stroke volume of the blood during that period is due, on the one hand, to an increased volume of the circulating blood, and on the other hand, to a response of the cardiovascular system directed toward a faster elimination of the oxygen debt. As a rule, the arterial pressure returned to the initial values rather quickly (after 5-15 minutes of the aftereffect).

Characteristic changes were observed in the indices of the respiratory system. As the magnitude of the acceleration increased owing to the action of the inertial forces, the resistance to inhalation increased, as was also confirmed by electromyographic data. Against a background of a general increase of the bioelectrical activity of the respiratory muscles, a periodic increase in activity coinciding with the inhalation phase was observed. The most distinct picture of this kind was observed at accelerations above 6 G.

Resistance to inhalation resulted in changes in the volume indices of external respiration. As is evident from the graph (Figure 4), the depth of breathing increased only up to an acceleration of 6 G. Greater accelerations led to a gradual decrease of this index, and at 20-22 G the depth of breathing in some of the subjects approached the volume of the dead air spaces (in two cases, less than 40 ml). The lung ventilation increased up to an acceleration of 10 G. At higher acceleration values, this index decreased gradually. Changes in the vital capacity of the lungs were of interest. In view of the difficulty involved in the testing, this index (with a few exceptions) was studied only up to an acceleration of 10 G. For greater reliability, in addition to the absolute value, an estimate was made of the ratio of the vital capacity of the lungs during the influence of acceleration to the background vital capacity in the same experiment. This ratio is shown on the graph. The higher the acceleration, the lower the vital capacity of the lungs.

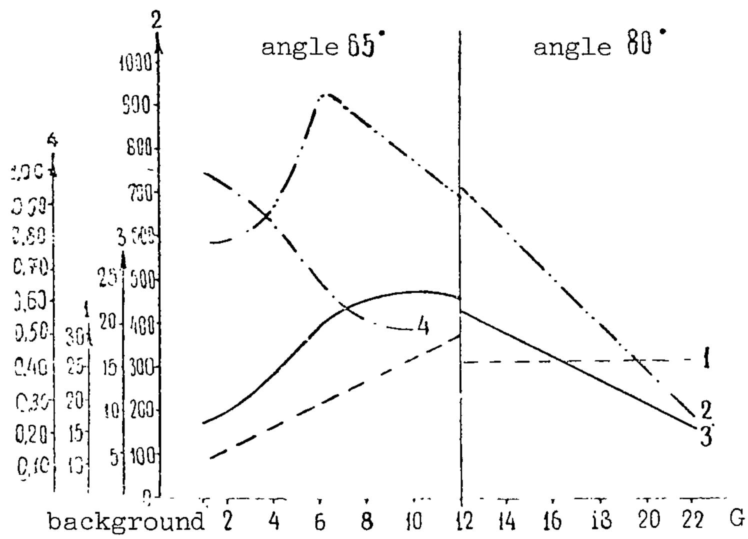

Figure 4. Average values of basic indices of external respiration in the range of accelerations studied:
1, breathing rate; 2, respiratory capacity; 3, respiratory capacity per minute; 4, vital capacity of lungs (at accelerations of 2 to 12 G in experiments with a 65° angle, at accelerations of 12 to 22 G in experiments with an 80° angle)

The X-ray data proved to be graphic from the standpoint of the estimate of the volume indices of external respiration. In experiments with an 80° angle, the area of the lung field at 2 G amounted to 95 percent of the initial value; at 4 G, to 93 percent; at 6 G, to 93 percent; at Hg, to 91 percent; at 12 G, to 79 percent; and experiments with acceleration at an angle of 65°, respectively, 97 percent, 97 percent, 94 percent, 93 percent, and 87 percent.

A definite regularity was also observed in the changes of the breathing frequency. In the range of accelerations from 4-12 G (65° angle), the breathing frequency changed linearly with the acting force (tangent of the slope, 2.8). At higher accelerations (80° angle), the breathing frequency remained approximately constant (25-27 respirations per minute).

A characteristic sequence was observed in the dynamics of the changes in the respiratory indices. The stage of the speedup in the velocity of the centrifuge was associated with an increase in respiratory frequency. The respiratory capacity and the ventilation of the lungs also increased, but although the acceleration exceeded 6-10 G, the depth of breathing and consequently the lung ventilation decreased. The beginning of the plateau was characterized by more or less pronounced variations of the indices being studied. Then, after a certain time which depended on the force of the acting factor and the individual circumstances, a stabilization of the new functional level of this system was observed.

During the period close to the subject's signal to stop the centrifuge, the respiration again became more frequent, and was accompanied by a decrease in the depth of breathing. As the acceleration increased during the slowing down of the centrifuge, there was observed a drop in the breathing frequency and a sharp increase in the respiratory capacity and lung ventilation. In the course of the following aftereffect (usually 15-20 min), the indices of external respiration gradually approached the initial level.

In evaluating the conditions of gas exchange in the lungs, we should not disregard the fact that during this type of interaction some impairments occur in the minor circulatory system (J. E. Herschgold, A. A. Kiselev, 1962, and others). The X-ray results also agree with this assumption. In all cases, we observed an increase in transparency typical of this type of acceleration, a depletion of the vascular pattern in the anterior sections of the pulmonary field, and a decrease of transparency in the posterobasal sections.

Thus, on the basis of an analysis of the changes of the cardiovascular system and system of external respiration, it is apparent that as the forces of the acting factor increase, considerable obstacles to the supply of oxygen arise in the organism. It is natural that against the background of a rising activity of a whole series of systems and organs, this should lead to the appearance of an oxygen debt which is cleared up only during the aftereffect. We evaluated the magnitude of the oxygen debt from the dynamics of the curve of the breathing capacity per minute and from the gas analysis of the exhaled air at various stages of the experiment.

We were able to determine the functional state of the central nervous system to some extent from the character of the electroencephalogram and the man's level of working capacity. According to the data of the EEG, all accelerations studied (12-22 G) may be divided into two groups. The first includes accelerations up to 14 G. During the period of increase in acceleration and during the first seconds of the plateau there occurred an increase in all the components of the EEG (Figure 5). During the fifth to the fifteenth seconds of the plateau the total potential of the slow components (1-8 c/s) underwent a relative decrease, but remained at a considerably higher level than in the initial state. At the end of the plateau, i.e., immediately before the subject's signal to stop the experiment, there again occurred an increase in the slow components, and this, in accordance with the existing concepts, may indicate the activity of the deceleration processes. It should be noted that at the same magnitudes of the accelerations in experiments with acceleration at a 65° angle, the increase in the slow components took place at the very beginning of the plateau and occasionally also at the end of the speedup period. It must be assumed that this is due to more profound shifts in hemodynamics, caused by a greater acceleration component along the pelvis-head axis.

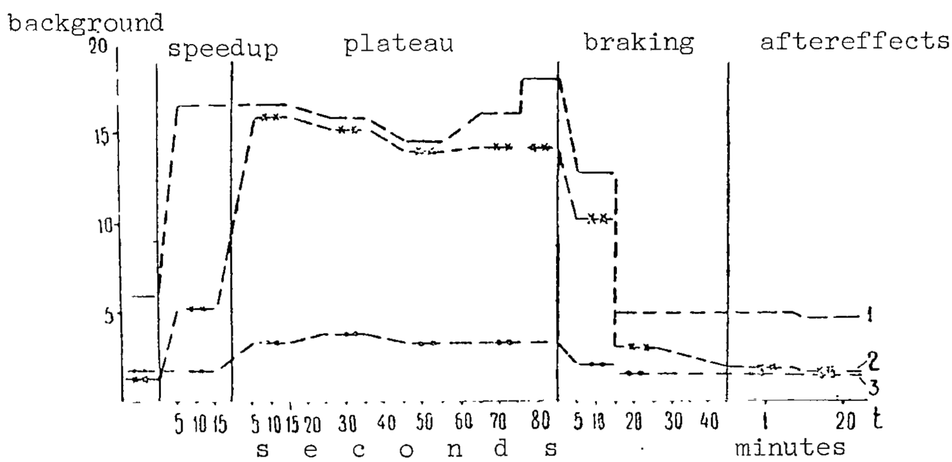

Figure 5. Variation in the integral values of the EEG components during the action of a 12 G acceleration at an 80° angle: 1, 1-8 c/s; 2, 13-30 c/s; 3, 8-13 c/s

The second group includes accelerations of 16-22 G. Here, as in the first case, an increase is observed in all the components of the EEG during the speedup period of the centrifuge and at the start of the plateau. However, no further decrease in the slow components occurred. Having reached their maximum during the 10-15th seconds of the plateau, they remained at this level with some fluctuations, until the very end of this period, and sometimes even until the first 2-7 sec of the deceleration. The period of the following aftereffect was characterized by an increase in the degree of exaltation of the α-rhythm in the test involving the closing of the eyes, and was distinctly expressed by a lengthening of the latent period of reaction to the opening of the eyes.

In evaluating the changes of the functional state of the central nervous system, we also took into account the fact that these shifts may be caused not only by known impairments in hemodynamics, but also by a higher afferent impulsation and the indirect effect of inertial forces on the brain tissues. However, the pronounced character of the hemodynamic shifts is indicated by the fact that during acceleration acting at a 65° angle, a complete loss of vision ("black shroud") occurred already at 10-12 G, whereas at 80° this phenomenon took place only at 16 G, and the acuity of binocular vision (average data) was 1.0 at 4-6 G, 0.8 at 8 G, 0.7 at 10 G, 0.4 at 12 G, and 0.2 at 14 G. The angle of binocular vision under the same conditions was 160° at 8 G, 60° at 10 G, and 10-14° at 12-14 G.

From the standpoint of an overall biological evaluation, of particular interest is an examination of the stages of development of the responses of the organism. Immediately before the application of acceleration, because of the emotional tension, there occurs a certain activation of a number of functional systems. During the speedup of the centrifuge associated with the steadily increasing acting force, this phenomenon becomes most pronounced. The lung ventilation increases, the frequency of the heartbeat and the arterial pressure rise, the bioelectrical activity of a number of organs and systems increases, etc.

There is evidence to indicate that this increase in activity is accompanied primarily by an impairment of the complex system of coordination of the functions and of the rhythmicity of a number of processes. In responses which are most closely related to the rhythmicity of the acting force, a decrease in this activity occurs when the inertial forces begin to manifest an appreciable resistance. Thus, for example, if the acceleration rises above 6-10 G, there occurs a steady drop of the depth of breathing against the background of a steadily increasing tension of the respiratory muscles. Other reactions such as the frequency of the heartbeat and breathing may continue their increase up to the time when the centrifuge reaches a steady state of operation (plateau).

After approximately 10-15 sec of this period, the following stage in the development of the responses of the organism takes place. This stage may be defined as the resistance stage, i.e., the stage of definite and established interrelationships between the organism and the new conditions of the ambient medium. It should be remembered, however, that the steady depletion of the energy level of the adaptation reactions takes place continuously during this period. In particular, this is related to a continuously increasing oxygen deficit.

If the effects of acceleration continue for a sufficiently long period, a third stage follows, which is characterized by a disruption of the adaptive reactions. For some indices, this is associated with a short-lived increase in their activity, and for others, with a decrease in activity. In high values of acceleration, this stage may be a direct continuation of the first period. In experiments with animals (A. S. Barer, I. M. Khazen, 1958), it was established that such a process of depletion also takes place in the deeper mechanisms of regulatory functions, in particular, the sympathetic-adrenal system. A relative and selective decrease in the level of noradrenalin takes place here while the level of adrenalin either remains the same or increases.

As the force decreases, or the action ends, the restoration period begins. In many systems and organs, this is associated with another increase in their activity (pulmonary ventilation, stroke volume of the heart, etc.). Gradually, after 10-20 minutes of the aftereffect, there takes place a complete normalization of all the external manifestations of the basic functional systems of the organism. For example, a function such as the working capacity differs very little from the initial level as early as the first few minutes of the aftereffect. It should be remembered, however, that many intimate aspects of vital activity may have a considerably longer period of aftereffects.

There is reason to believe that the prevailing processes take place in accordance with the principles of the theory of automatic control. From this standpoint, the second and fourth stage may be characterized as transitional functions.

In conclusion, we can state that the set of investigations which we carried out, combined with the literature sources, makes it possible, on the one hand, to determine certain means of raising man's resistance to the prolonged action of accelerations and to establish the endurance limits and, on the other hand, by identifying a series of regularities in the response reactions of the organism to this action, to formulate the problems requiring solutions. This applies primarily to a deeper analysis of the intimate aspects of vital activity, which will undoubtedly indicate new ways of solving one of the most pressing problems in present-day aerospace physiology.

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